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LEAD POISONING IN THE MINING OF LEAD IN UTAH

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By Arthur L. Murray

INTRODUCTION

Lead poisoning, or plumbism, is one of the oldest known forms of metallic poisoning. As early as the first century of the Christian Era Pliny the Younger mentions plumbism as one of the recognized diseases among slaves who worked in mines. Nearly a hundred years earlier the application of the metal to commercial use had demonstrated that lead poisoning might be a menace to the public.

Pipes of beaten lead were introduced by Roman builders to convey water for domestic use from masonry aqueducts to houses. Several severe outbreaks of colica pictonum, better known to us to-day as lead poisoning, resulted. The ability of water to attack metallic lead and convey to the human system the poisonous properties derived from the metal varies in proportion to the plumbo-solvency inherent to the water. The plumbo-solvency of a given water supply may differ at different periods; thus, a water which under normal conditions is a very poor solvent of lead, when acidulated through contamination with such agencies as humic or ulmic ground acids, acid-forming bacteria, an excess of oxygen or carbon dioxide, nitrates or chlorides, may develop a much higher solvency. After serious outbreaks of lead poisoning, due to changed water conditions that increased the lead solvency of water supplied to several Roman communities, the danger of using lead pipes to convey water used for domestic purposes was recognized. Vitruvius, the famous Roman architect and engineer, publicly condemned the use of lead for piping water for domestic use. As far as the author knows, these are the earliest instances on record of lead being recognized as a source of poisoning.

To-day, lead is known to be the most widely distributed cause of all the metallic poisonings, and it is recognized as a hazard of many occupations. In one form or another lead is used in 138 industries; certain of these use lead in a form or in a process which affords only a slight opportunity for poisoning, but many of them use lead or lead salts in processes which present decided hazards to the workmen.

Foremost among the industries in which lead poisoning is a recognized hazard are the following: Lead mining, smelting, and refining; grinding and mixing lead paints; painting, glazing, and decorating white wear, pottery, tiles, and porcelain; typefounding and typesetting; manufacture and repair of storage batteries; and the manufacture of rubber goods.

TOXIC FORMS OF LEAD

Lead used in industry enters the body in the form of a dust. Lead dust, in most cases of industrial lead poisoning, occurs as a lead salt; but in certain occupations, such as file cutting, precious stone grinding, and the filing of lead castings, finely divided particles of metallic lead may be taken into the body.

Lead and its more common salts used in industry rank as follows, according to their solubility in the fluids of the body: Acetate, chloride, nitrate, carbonate, white oxide, red oxide, sulphate, sulphide, and metallic lead. Until comparatively recent years many writers considered the sulphate and sulphide of lead to be of little or no consequence in the causation of lead poisoning, but two very interesting series of tests made in this country demonstrated beyond a doubt that basic lead sulphate and lead sulphide and the dust from lead sulphide ore are all soluble in the human gastric juice at body temperature.

In the first series of tests [1] basic lead carbonate and basic lead sulphate derived from paint dusts, dry samples of sublimed white lead (basic lead sulphate), and dry samples of lead carbonate ("old Dutch process") were used. Determined as sulphate these several samples contained the following averages in grams of lead sulphate per gram of dust or dry sample:

	Grams
Basic lead carbonate, paint dust	1.05
Basic lead sulphate, paint dust	.85
Lead carbonate, dry	1.12
Basic lead sulphate, dry	.97

Normal gastric juice was obtained from a man 27 years of age who had a complete constriction of the œsophagus and a gastric fistula of 16 years' standing. The gastric juice was secreted while he was chewing palatable food when hungry, thus producing normal "appetite" gastric juice not mixed with saliva.

Four tests, in which 0.5 gram of basic lead sulphate, paint dust, mixed with 25 c. c. of water was digested at 38° C. (100.4° F.) for

[1] Carlson, A. J., and Woelfel, A., Solubility of Basic Lead Sulphate and Basic Lead Carbonate in Human Gastric Juice and Relative Toxicity of the Two Salts as shown by Feeding Experiments with Dogs and Cats: Bull. 120, U. S. Bureau of Labor Statistics, 1913, p. 22 et seq.

10 hours in 25 c. c. of gastric juice, gave on eight analyses an average solubility of 0.0473 gram or 9.5 per cent.

Three similar tests made with basic lead carbonate, paint dust, gave on six analyses an average solubility of 0.2304 gram or 46.1 per cent.

The results from the tests made with dry lead sulphate and dry lead carbonate are given in the following table:

Relative solubility of basic lead sulphate (sublimed white lead) and lead carbonate ("old Dutch process") in human gastric juice

Lead sulphate tests			Lead carbonate tests		
Experiment No.	Digestive mixture	Lead dissolved, gram	Experiment No.	Digestive mixture	Lead dissolved, gram
1	25 c. c. gastric juice; 25 c. c. water; 0.5 gram lead sulphate, at 38° C. (100.4° F.) for 10 hours.	[1] 0.1260 [2] .1210	1	25 c. c. gastric juice; 25 c. c. water; 0.5 gram lead carbonate, at 38° C. (100.4° F.) for 10 hours.	[1] 0.2940 [2] .3044
2	25 c. c. gastric juice; 25 c. c. water; 0.5 gram lead sulphate; 0.1 gram peptone (a), 0.5 gram peptone (b), at 38° C. (100.4° F.) for 10 hours.	[3] .1376 [4] .1284	2	25 c. c. gastric juice; 25 c. c. water; 0.5 gram lead carbonate; 0.1 gram peptone (a), 0.5 gram peptone (b), at 38° C. (100.4° F.) for 10 hours.	[3] .3302 [4] .3100
3	50 c. c. gastric juice; 0.5 gram lead sulphate at 38° C. (100.4° F.) for 10 hours	.1500	3	50 c. c. gastric juice; 0.5 gram lead carbonate at 38° C. (100.4° F.) for 10 hours	.3896
		Gram / *Per cent*			*Gram* / *Per cent*
Average	1	0.1235 / 24.7	Average	1	0.2992 / 59.8
	2	.1330 / 26.6		2	.3201 / 64.0
	3	.1500 / 30.0		3	.3896 / 77.9

[1] Test 1.
[2] Test 2 (check).
[3] Digested with 0.1 gram peptone.
[4] Digested with 0.5 gram peptone.

In the author's opinion the findings from these tests prove, far more conclusively than feeding experiments with animals, that lead sulphate is soluble in human gastric juice and can be a causative agent in producing lead poisoning. As shown in the previous table, the ratio of the solubility of the sulphate in the gastric juice compared to the solubility of the carbonate is relatively 1 to 2½.

The second series of tests [2] made with lead sulphide and lead sulphide ore dust to determine their solubility in human gastric juice were as follows:

Gastric juice was taken from the same subject and under the same conditions as that used in the previous series of tests. Samples of lead sulphide ore dusts were obtained from the Flat River and Desloge concentrators of the Federal Lead Co., and from the Bonne Terre concentrator of the St. Joseph Lead Co. These samples, ac-

[2] Woefel, A., and Carlson, A. J., The Solubility of Lead Sulphide Ores and of Lead Sulphide in Human Gastric Juice: Bull. 141, U. S. Bureau of Labor Statistics, 1914, p. 82 et seq.

cording to assays furnished by the companies, gave the following percentages of lead:

Percentages of lead in samples of lead dusts

	Per cent
Federal Lead Co.:	
Flat River concentrator	73.4
Desloge concentrator	70.1
St. Joseph Lead Co. Bonne Terre concentrator	72.0

Together with the foregoing samples parallel tests were run with a laboratory sample of lead sulphide. The procedure in these tests was much like that cited above, and consisted of digesting 0.5 gram of lead sulphide ore dust and a laboratory sample of lead sulphide with 25 cubic centimeters of water in 25 cubic centimeters of gastric juice at 38° C. (100.4° F.) for 10 hours. The results obtained are shown in the next table.

Relative solubility of samples of lead sulphide in human gastric juice

Samples	Number of tests	Amount of lead sulphide ore used	Lead contained in ore	Lead sulphate derived from digestion	Lead contained in lead sulphate	Average lead dissolved
		Gram	*Gram*	*Gram*	*Gram*	*Per cent*
Flat River concentrator	1	0.5	0.367	0.0164	0.0111	
	2	.5	.367	.0156	.0106	2.94
	3	.5	.367	.0158	.0107	
Desloge concentrator	1	.5	.350	.0072	.0049	
	2	.5	.350	.0076	.0051	1.38
	3	.5	.350	.0066	.0045	
Bonne Terre concentrator	1	.5	.360	.0172	.0117	
	2	.5	.360	.0178	.0121	3.32
	3	.5	.360	.0176	.0120	
Laboratory, lead sulphide	1	.5	.433	.027	.0185	
	2	.5	.433	.029	.0197	4.60
	3	.5	.433	.032	.0218	

The foregoing series of tests shows that lead sulphide, in both the pure state and as a dust from lead sulphide ores, is soluble in human gastric juice at body temperature. The solubility of lead sulphide in human gastric juice is considerably less than that of lead carbonate and lead sulphate, as shown by the table below, but it is still soluble enough to be dangerous to the health of persons engaged in mining and milling lead sulphide ores when the processes used cause the production of fine dry dust.

Relative solubility of lead carbonate, lead sulphate, and lead sulphide in human gastric juice at body temperature

	Per cent of solubility
Lead carbonate	46.1
Lead sulphate	9.5
Lead sulphide	2.5

The author believes that the reason that lead sulphide has not been more generally recognized as a source of danger in the causation of lead poisoning is not so much because of its low solubility as because of certain physical characteristics of the dust from that

particular salt of lead. These characteristics will be discussed later in connection with mining.

Metallic lead, except in connection with a few restricted industries, is rarely taken into the human system. Metallic lead dust has such a high specific gravity that unless it is extremely fine it is not held in suspension in the air for any considerable length of time and hence there is small opportunity for it to be inhaled. Several cases of lead poisoning caused by leaden bullets retained within the body are on record.

CHANNELS BY WHICH LEAD ENTERS THE BODY

Lead normally enters the system by only three channels. The exception to this statement is that metallic lead or salts of lead are sometimes forced into the body through injuries, accidental or intentional. In the order of their importance and relative frequency, these three channels of entrance are through inhaled air, through the mouth by direct swallowing, and through the skin. These three channels will be discussed in the reverse order of their frequency and importance.

Under extremely favorable circumstances lead may enter the body through the skin. To do so the lead must be in a very soluble form and the skin in a receptive state. Authentic cases of lead poisoning through the skin are rare and almost wholly confined to poisonings which follow the use of certain cosmetics or hair dyes.

The mouth and alimentary canal, through the direct swallowing of food contaminated with lead or lead salts, the drinking of leaded water, and the chewing of tobacco soiled by lead, is the next most frequent way by which lead enters the body.

Investigations carried on both abroad and in this country tend to prove that the greater part, or virtually all, of the industrial lead poisoning is due to lead taken into the system through inhaled air. Besides the weight of findings obtained through investigations, it is obvious that air affords the greatest opportunity for lead to enter the body. Lead and the salts of lead are used or handled extensively in many industries. All the salts of lead and the metal itself are capable of subdivision into fine dust. The processes under which they are handled frequently cause lead dust to be formed and escape into the atmosphere in which employees are working, so that the air breathed by the workers offers an ever-present medium for lead dust to be carried into the body.

Although inhaled air is its medium for entrance, inhaled lead dust is absorbed chiefly through the alimentary system and only to a moderate degree through the respiratory tract. Experiments [3] have

[3] Kober, George M., and Hanson, Wm. C., Diseases of Occupation and Vocational Hygiene. Philadelphia, 1916, p. 83.

proved that when lead dust is inhaled, only 12 per cent reaches the lungs and that 70 per cent finds its way into the alimentary canal. On entering the nostrils particles of the dust, especially the larger particles, impinge upon the moist hairs and mucous membrane of the lower nasal passages, from whence it is expelled with the usual nasal excretions. Most of the remaining dust is sifted out of the breathed air in the nasopharynx, where it combines with mucous secretions and is swallowed.

The swallowed lead dust after passing into the stomach is acted upon by the gastric juice. The rate of absorption depends upon the nature of the lead salt from which the dust is derived.

Data based upon scientific experimentation dealing with the effect of lead dust entering the lungs have been almost a minus quantity until recently. As early as 1839 Tanquerel des Planches [4] demonstrated that lead insufflated by a dog through a tracheal cannula could be absorbed into the system. No confirmatory work seems to have been undertaken until Blumgart,[5] several years ago, demonstrated with dogs, so prepared that lead could not enter either the respiratory or gastrointestinal tract, that lead carbonate could be absorbed into the system from the nasopharynx.

The more recent experiments of Minot,[6] in which cats especially prepared by careful aseptic ligation of the œsophagus received lead salts suspended in sterile physiological salt solution either through small tubes inserted into the trachea or by direct injection into the lumen of the trachea, showed conclusively that after its insufflation lead can readily be absorbed by these animals through their lungs. By thus closing off through the ligation of the œsophagus all the channels by which lead might reach the gastrointestinal tract, absorption could only take place through the lungs and the mucous membrane of the respiratory tract.

The lead salts used in the experiments with animals were as follows: Lead carbonate with 15 animals, lead chromate with 2 animals, natural lead sulphide (galena) with 3 animals, and lead oxide with 5 animals. The duration of exposure ranged from 12 to 162 hours; in every instance post-mortem analyses of the body tissues proved marked absorption of lead through the lungs. One of the striking features of these experiments is that natural lead sulphide (galena) seems to be as readily absorbable through the lungs as either the carbonate or oxide of lead.

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[4] Tanquerel des Planches, L., Traite des Maladies de Plomb ou Saturnines. T. 1, Paris, 1839, p. 88.

[5] Blumgart, H. L., "Lead studies, VI": Jour. Ind. Hygiene, vol 5, 1923–24, p. 153.

[6] Minot, A. S., Lead Studies, V. B., "The distribution of lead in the organism after absorption by the lungs and subcutaneous tissue": Jour. Ind. Hygiene, vol. 6, 1924–25, p. 137 et seq.

OCCURRENCE OF LEAD POISONING IN MINING

Many excellent articles dealing with the occurrence of industrial lead poisoning in this country have been published in recent years; notable among these are the articles by Alice Hamilton [7] and John B. Andrews.[8] Although articles bearing upon the subject of lead poisoning are numerous and embrace nearly every industry, including the metallurgy of lead, in which lead is utilized to any extent, there are almost no writings, with the exception of short sections appearing in textbooks, which have presented the hazards of lead poisoning in the mining of lead ores.

Data and statistics dealing with the occurrence of lead poisoning in the mining of these ores are rare and incomplete, and as stated before, few of the authorities who have written on lead poisoning refer to the lead hazard in mining.[9]

The records of the Hospital of St. Vincent in Leadville, Colo., furnish some data on the occurrence of lead poisoning in mining, as reported by Dr. Alice Hamilton in Lead Poisoning in the United States.[10] In 1880 that hospital treated 121 cases of lead poisoning; of these cases 67 were miners and 54 were smelter men. The following year 160 cases were treated, of which 41 were miners and 119 were smelter employees. In 1882 the total cases of lead poisoning treated were 478, of which 54 were miners and 424 were men who worked in smelters. For some years following the number of miners with lead poisoning treated at this hospital ranged between 40 and 50, but as the mines went deeper and the ore changed from the carbonate and oxide to the sulphide the cases of lead poisoning among the miners began to decrease; by 1910 there were but 8 cases treated in the year; in 1911, 3; and for 1912 there were no cases reported.

The same author [11] reports that while investigating lead poisoning among smelter employees in and about St. Louis records were found of 25 cases of lead poisoning treated in one hospital, all of which

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[7] Hamilton, Alice, The White Lead Industry in the United States, with an appendix on the Lead-Oxide Industry: Bull. 95, U. S. Bureau of Labor Statistics, July, 1911, pp. 189–259. Lead Poisoning in Potteries, Tile Works, and Porcelain Enameled Sanitary Ware Factories: Bull. 104, U. S. Bureau of Labor Statistics, August, 1912, pp. 1–82. Hygiene of the Painters' Trade: Bull. 120, U. S. Bureau of Labor Statistics, May, 1913, pp. 1–66. Lead Poisoning in the Smelting and Refining of Lead: Bull. 141, U. S. Bureau of Labor Statistics, February, 1914, pp. 1–81. Lead Poisoning in the Manufacture of Storage Batteries: Bull 165, U. S. Bureau of Labor Statistics, December, 1914, pp. 1–34.

[8] Andrews, John B., Deaths from Lead Poisoning in New York State in 1909 and 1910: Bull. 95, U. S. Bureau of Labor Statistics, July, 1911, pp. 260–282.

[9] Hoffman, Frederick L., Mortality from Respiratory Diseases in Dusty Trades: Bull. 231, U. S. Bureau of Labor Statistics, June, 1918, p. 356 et seq. Oliver, Thomas, Industrial Lead Poisoning with a Description of Lead Processes in Certain Industries in Great Britain and the Western States of Europe: Bull. 95, U. S. Bureau of Labor Statistics, July, 1911, pp. 1–142.

[10] Kober, George M., and Hanson, W. C., Diseases of Occupation and Vocational Hygiene. Philadelphia, 1916, p. 113.

[11] Hamilton, Alice, Lead Poisoning in the Smelting and Refining of Lead: Bull. 141, U. S. Bureau of Labor Statistics, February, 1914, p. 17 et seq.

cases were traced to the mines and concentrating mills of the Bonne Terre and Flat River lead districts.

Certain data pertaining to the occurrence of lead poisoning among a class of smelter employees are of interest and reflect the danger to those engaged in mining lead ores. The unloading gangs who dump ore from cars received at the smelters are subjected to very much the same types of ore dusts as the miners are, but generally in a less confined space than the miners, for most smelter ore bins are in semiopen structures.

The following information is taken from Lead Poisoning in the Smelting and Refining of Lead,[12] and shows the record of lead poisoning for a yard gang engaged in unloading, wheeling charges, etc.:

Number of employees examined	26
Number of cases of plumbism	5
Rates per 1,000 employees	19.2

From another smelter a 2-month record of lead poisoning among their employees in the ore-bin department showed:

Number of employees	45
Number of cases of plumbism	6
Rate per 1,000 employees	13.3

LEAD POISONING IN UTAH IN RELATION TO MINING

The author was assigned to study and make a report on lead poisoning in the mines of Utah. For the past 30 years the State of Utah has ranked high among the lead-producing regions in the United States. Its production for the period from 1890 to 1920 averaged 56,714 short tons per year; thus Utah presents an ample opportunity for study.

Visits to the principal mining districts of the State and conversations with mine superintendents, miners, and physicians serving the several camps elicited the information that lead poisoning had been and was still prevalent, but when statistical data were sought no source could be found from which the relative frequency of cases for the State could be obtained. Utah has no statute or ordinance requiring the reporting of occupational diseases.

A study of the records of the Utah State Board of Health furnished data on deaths from lead poisoning, as shown in the following statement:

	Deaths		Deaths
1910	1	1917	3
1911	0	1918	1
1912	2	1919	2
1913	1	1920	2
1914	6	1921	2
1915	4	1922	1
1916	1		

[12] Hamilton, Alice, work cited, p. 66 et seq.

The few deaths seem to indicate a fairly low case rate, but it should be borne in mind that lead poisoning is rarely an immediate cause of death, and that the train of sequelæ following acute attacks leads to chronic involvements of the digestive, nervous, circulatory, or genito-urinary systems; these involvements manifest themselves months and even years after the acute symptoms of lead poisoning have disappeared. The time elapsing between the presence of active symptoms of lead poisoning and the terminal illness from pathological conditions excited by lead absorption is so long that the initial cause—lead poisoning—is frequently lost sight of entirely and the death certificate gives no evidence of the primary cause.

EXTENT OF POISONING THROUGHOUT STATE

With a view to obtaining first-hand information on the extent of lead poisoning throughout the State, and especially the distribution of cases, the physicians and dentists of Utah were requested to cooperate in an arrangement whereby they would report all cases of lead poisoning encountered or treated during the years 1919 and 1920. Blanks for reporting cases by initials and ages were furnished them.

The cooperation obtained was most cordial; 256 practitioners, or about 65 per cent of the active physicians and dentists of the State, submitted returns. Acknowledgment is made of the assistance rendered by the physicians and dentists of Utah in making possible this phase of the investigation. Undoubtedly certain physicians and dentists who did not make returns failed to come in contact with cases of lead poisoning and thus had nothing to report. The physicians reported 349 cases for 1919 and 218 cases for 1920; the dentists reported 22 cases for 1919 and 6 cases for 1920.

The returns covered nearly every section of the State large enough to support a physician or a dentist. The aggregate population of those sections from which returns were received equaled about 70 per cent of that of the entire State.

An analysis of the returns furnished some interesting findings. The 371 cases reported for 1919 and 224 cases for 1920 gave lead poisoning rates of 0.83 and 0.50 per 1,000 for the respective years for the entire population of the State. These rates are strikingly high for a State in which the principal industries are agriculture and stock raising.

A study of the cases in the light of the character of the communities from which they were reported affords data of particular interest. Returns were received from 57 different sections. The cases, grouped according to communities of like character, with the

percentage of cases of lead poisoning reported for each group to the total cases reported, are tabulated as follows:

Comparative percentages of the frequency of lead poisoning in different communities in Utah

Number of communities	Character of community	Cases of lead poisoning reported		Percentage of total cases reported	
		1919	1920	1919	1920
38	Agriculture, stock raising, fruit growing, and dairying	14	11	3.8	4.9
1	Cement works	0	0	0	0
6	Metal mining	264	184	71.2	82.1
5	Concentrating and smelting	39	16	10.5	7.1
1	Railroad	0	0	0	0
3	Medical center	54	13	14.5	5.9
3	Coal mining	0	0	0	0

The number of cases of lead poisoning reported for both years from the agricultural, stock-raising, fruit-growing, and dairying sections of the State hardly seems compatible with the chief industries of those sections. A study of those sections and of the cases reported from each community shows that in 1919, 9 out of the 14 cases reported, and in 1920, 7 out of the 11 cases reported, were from agricultural, stock-raising, fruit-growing, and dairying sections adjacent to mining or smelter districts. In these districts many of the men who follow farming during the summer and fall seek employment in the mines and smelters during the slack months of the winter and spring. A physician and a dentist whose combined reports totaled 10 cases for 1919, and 5 cases for 1920, from agriculture communities, said that most of their patients had contracted lead poisoning in the mines. It is a common practice for men who have contracted lead poisoning in mines and smelters to seek outdoor employment on farms and ranches in the hope of regaining health.

The cases reported for the medical centers are believed to be mostly chronic or aggravated cases of lead poisoning sent from the mines and smelters to the medical centers for treatment in hospitals or by specialists. One specialist reported that the number of cases referred to him from mines averaged two a month.

The smelter cases, considering the known exposure of smelter employees to lead, shows a case rate that is very low.

Case rate per 100,000 shifts worked and per 1,000 employees in the smelters of Utah for 1919 and 1920

Year	Shifts worked	Cases of lead poisoning reported	Cases per 100,000 shifts	Cases per 1,000 employees
1919	921,527	39	4.2	15
1920	808,175	16	1.9	7

The foregoing rates compare favorably with those of other lead smelters throughout the United States.

The metal-mining camps of Utah supplied 71.2 per cent of all the lead-poisoning cases reported in 1919, and 82.1 per cent of the cases reported in 1920. These figures do not take into consideration the cases of lead poisoning sent to medical centers for treatment, or the cases reported from agricultural sections. It is probable that if all the cases could be traced to the places where they were contracted the metal mines would prove to be accountable for approximately 90 per cent of all the cases of lead poisoning in Utah.

CASES OF POISONING IN MINING CAMPS

Because of the convincing evidence that there was in Utah a decided relation between the occurrence of lead poisoning and the mining of lead ores, a canvass was made of the chief metal-mining camps of the State to obtain records of the cases of lead poisoning contracted during a period of some years. In only one camp could records of any consequence be found. At Park City, Utah, a hospital that cared for most of the surgical cases and the more serious medical cases of the camp had been maintained from October, 1904, to April, 1919, when it was closed for lack of funds. This was the Miners' Union Hospital, conducted under the direction of the Park City local of the miners' union, to whom the author is indebted for the privilege of making a study of the records of the cases treated in the hospital.

Number of cases admitted to the Miners' Union Hospital, Park City, Utah, showing the cases of lead poisoning and probable cases of lead poisoning for the period, October, 1904, to April, 1919

Year	Total cases admitted	Cases of lead poisoning	Probable cases of lead poisoning—stomach trouble, bowel trouble, and cramps	Year	Total cases admitted	Cases of lead poisoning	Probable cases of lead poisoning—stomach trouble, bowel trouble, and cramps
1904 [1]	54	8	0	1913	165	20	1
1905	191	17	1	1914	166	15	1
1906	207	9	6	1915	127	8	1
1907	224	6	10	1916	219	25	0
1908	93	1	14	1917	181	9	6
1909	202	13	10	1918	147	2	2
1910	239	19	18	1919 [2]	35	2	0
1911	356	36	13				
1912	196	34	0		2,702	224	102

[1] 1904, October, November, and December.
[2] 1919, January, February, March, and April.

A number of cases merely diagnosed as stomach trouble, bowel trouble, and cramps are shown in the table as probable cases of lead poisoning. One of the physicians who served as a member of the

staff of the hospital advised the author that, in his opinion, most of the cases entered as stomach trouble, bowel trouble, and cramps were mild cases of lead poisoning, but that in the absence of positive symptoms of lead poisoning they were not so designated. The cases of lead poisoning shown probably were not all that occurred in this camp as, because of its limited accommodations, only the most severe cases were placed in the hospital.

The 2,702 admissions during the period covered compared with the 224 cases of lead poisoning show that 8.3 per cent of all cases admitted to the hospital were due to lead. The probable cases of lead poisoning amounted to 3.7 per cent of the total admissions.

The arrangement under which the Miners' Union Hospital was conducted required the operating companies of Park City to pay each month a stated sum per employee. This payment entitled employees to hospital care when they required it. The records of monthly payments to the local were kindly furnished and for those years which were complete, 1905 to 1915, inclusive, the number of men employed each year in the mines of the district was determined. The following table shows the yearly rates per 1,000 employees for reported cases of lead poisoning and for probable cases of lead poisoning.

Yearly rates per 1,000 employees, Park City mining district, for reported cases and probable cases of lead poisoning, 1905 to 1915, inclusive

Year	Employees	Reported cases of lead poisoning	Rate per 1,000 employees	Probable cases of lead poisoning	Rate per 1,000 employees
1905	1,084	17	15.6	1	0.9
1906	1,197	9	7.5	6	5.0
1907	1,221	6	4.9	10	8.2
1908	611	1	1.6	14	22.9
1909	845	13	15.3	29	34.3
1910	919	19	20.7	18	19.6
1911	1,010	36	35.6	13	12.9
1912	956	34	35.6		
1913	1,063	20	18.8	1	.9
1914	901	15	16.6	1	1.1
1915	752	8	10.7	1	1.3

The yearly rates for both the reported cases of lead poisoning and for the probable cases of lead poisoning show wide variation. With but three exceptions, however, the yearly rates for reported cases of lead poisoning are more than 10 per 1,000 and for the period they average 16.6 cases per 1,000 employees. The reported cases and probable cases combined give an average yearly rate of 25.7 cases per 1,000 employees. There is no evident relation between the number of men employed and the cases and probable cases of lead poisoning.

A study of the tonnage and the lead content of the ore produced in the mines of the Park City district, and the rates per 1,000 em-

ployees for reported and probable cases of lead poisoning, offers some comparisons.

Comparison of the frequency of lead poisoning with the quantity of ore produced for the Park City mining district, 1905 to 1915, inclusive

Year	Employees	Rate per 1,000 employees		Ore production, short tons [1]	Lead content of ore, pounds [1]
		Reported cases of lead poisoning	Probable cases of lead poisoning		
1905	1,084	15.6	0.9	228,142	45,280,817
1906	1,197	7.5	5.0	264,792	46,511,176
1907	1,221	4.9	8.2	235,628	36,234,757
1908	611	1.6	22.9	142,331	34,051,699
1909	845	15.3	34.3	196,172	46,350,390
1910	919	20.7	19.6	215,339	38,129,761
1911	1,010	35.6	12.9	296,350	47,637,642
1912	956	35.6		280,671	42,111,561
1913	1,063	18.8	0.9	270,527	41,808,713
1914	901	16.6	1.1	236,952	32,323,066
1915	752	10.7	1.3	263,342	49,350,377

[1] From U. S. Geological Survey Professional Paper 111.

The table shows the natural tendency of the mine production to fluctuate with the number of men employed, although the ratio is not constant. The rates per 1,000 employees for reported cases and probable cases of lead poisoning show little relation to the amounts of ore produced or the lead content of the ore, where these two factors are considered by themselves.

For 8 out of the 11 years, however, there is a decided relation between the case rates and the tonnage per man and the lead content of the ore produced. This relation is what naturally would be expected. A decided increase or decrease in the tonnage mined per man, or an increase or decrease in the lead content of the ore handled would, in a general way, subject those employed to a greater or less exposure to lead absorption.

A comparison between the rates for reported and for probable cases of lead poisoning, the ore mined per man, and the lead content of the ore produced in the Park City mining district, for the years 1905 to 1915, inclusive, follows:

Year	Ore tonnage per man	Lead content of ore, per cent	Rate per 1,000 employees		Year	Ore tonnage per man	Lead content of ore, per cent	Rate per 1,000 employees	
			Reported cases of lead poisoning	Probable cases of lead poisoning				Reported cases of lead poisoning	Probable cases of lead poisoning
1905	210.4	9.9	15.6	0.9	1911	293.4	8.0	35.6	12.9
1906	221.2	8.8	7.5	5.0	1912	293.5	7.5	35.6	
1907	192.9	7.7	4.9	8.2	1913	254.4	7.7	18.8	.9
1908	232.9	11.9	1.6	22.9	1914	262.9	6.8	16.6	1.1
1909	232.1	11.8	15.3	34.3	1915	350.1	9.3	10.7	1.3
1910	234.3	8.8	20.7	19.6					

The foregoing table can only give a rough indication of the relation of lead poisoning to the lead ore mined, because of the variety of factors affecting both the cases reported and the exposure to lead hazards.

When considered by year periods, the high labor turnover so common to metal mines tends to lessen the cases reported. It is a common occurrence for men who are capable of recognizing the early symptoms of leading to move to mines free from lead or to those with less lead hazards.

In the mines the changing conditions underground during a period of 12 months may likewise have a decided bearing on the lead hazard to miners, yet the tonnage per man and the lead content of the ore mined may change but slightly. Shifting from dry to wet sections or from carbonate to sulphide zones would have a marked influence on the exposure to lead, as will be shown later.

There are 94 recognized mining districts in Utah distributed throughout 21 counties. Most of these districts are either abandoned or so small as to be of no consequence. A compilation of the cases of lead poisoning reported during the survey for the years 1919 and 1920 showed that all the cases reported for metal mines occurred in five of the districts. These districts, as would be expected, are the chief lead producers of the State. Through the cooperation of the physicians of these five districts the cases of lead poisoning for the years 1921, 1922, and 1923 have been obtained to supplement those for 1919 and 1920.

Number of cases of lead poisoning and the quantity of lead ores produced in the chief lead-producing districts of Utah for the years 1919 to 1923, inclusive

District	Cases of lead poisoning					Quantity of lead ores produced (short tons)				
	1919	1920	1921	1922	1923	1919	1920	1921	1922	1923
Bingham	2	0	2	2	1	109,785	176,966	157,967	91,223	147,343
Frisco	16	18	4	3	2	32,255	13,805	5,805	9,768	14,944
Ophir	7	2	2	0	1	64,302	3,053	2,687	55,135	60,379
Park City	81	14	33	23	27	86,350	128,435	101,073	182,765	193,693
Tintic	158	150	51	44	35	118,276	162,522	106,866	147,334	202,178
Total	264	184	92	72	66	410,968	484,781	374,898	486,225	618,537

In general, the districts given in the preceding table show a decided decline in cases of lead poisoning for the period covered. This decline, although not constant by years, tends to be regular. It shows little relation to the production of lead ores except that in many instances the cases have decreased as the production of lead ores has increased.

In the absence of definite information (data not available) showing the nature of the lead ores mined, the lead content of the ores, and the sections of the mines (dry or wet) from which ores were taken, an intelligent analysis of these figures can not be made. Information gathered from superintendents of the mines and the State mine inspector and the author's personal knowledge of the districts lead to the belief that since 1919 there has been a decided and constant increase in the sulphide content of the ores mined and that, at the largest producers especially, the ores have been taken from greater and greater depths, where the working places have been increasingly wet.

Approximately all cases of lead poisoning due to mining in Utah are contracted in the districts mentioned; based on the cases reported for these districts the yearly case rates per 1,000 for all men engaged in metal mining in the State are as follows:

Reported cases yearly of lead poisoning per 1,000 men employed in Utah metal mines

	Rate per 1,000 men		Rate per 1,000 men
1919	62.7	1922	17.7
1920	34.7	1923	10.0
1921	21.7		

A factor no doubt in reducing the number of cases of lead poisoning was a campaign to secure wet drilling and the sprinkling of muck piles before loading, conducted throughout the metal mines of the State by the Utah State metal mine inspection department. This campaign was started in the spring of 1920 and was continued until the close of 1924; at that time it was estimated that more than 95 per cent of all drilling machines used underground in the metal mines of Utah were of the wet type. During 1920 and 1921 efforts were centered on replacing the jackhammers, during the following year the drifting machines were replaced, and during 1923 the stopers were replaced.

It may be only a coincidence, but as the campaign for wet drilling and wetting down muck piles progressed the most marked decline in lead poisoning took place. This decline is shown by the case rates for the successive years of the campaign, as given in the table printed above.

The improvement in 1920 and later years represents the results of the campaign for the replacement of dry drills by wet drills and the wetting down of muck piles.

MINING CONDITIONS AND CHARACTER OF ORES PREDISPOSING TO LEAD POISONING

The conditions under which lead ore is mined and the nature of the ore have a direct bearing on the incidence of lead poisoning among miners.

The stage of development of mines working large deposits of lead ore often influences the prevalence of lead poisoning in such mines. Generally speaking, in Utah mines the more soluble salts of lead occur close to the surface and with few exceptions the upper levels of the mines are dry and dusty; therefore, in the earlier stages of the development of the mine, when the work is confined to a few hundred feet below the surface, the lead hazard is greatest.

The situation of mine openings and the efficiency of the mine ventilation play important parts in exposure to lead poisoning. Where the working faces are distant from good air courses, or where for any reason the movement of air at the working face is sluggish, the lead dust thrown into the air through drilling, blasting, and mucking, remains suspended in the atmosphere and is breathed by the miners for considerably longer periods of time than it would be if the ventilation at the faces was good, which at the outset would cause greater dilution of the dust in the air and tend to remove dust-laden air from the working face with greater rapidity.

Heat and humidity may also cause predisposition to lead poisoning. In those sections of mines where the temperature is high and the humidity great, men wear little clothing and even strip to the waist, thus exposing a large part of the body to lead-laden dust. Men working under these conditions usually perspire profusely, and thus are more likely to absorb soluble salts of lead through the skin. Then, too, unless they bathe after each shift the lead dust accumulated on their bodies becomes dry and they are liable to inhale lead dust while changing clothes and in sleeping quarters.

The method of drilling and handling muck may be a decided predisposing factor in causing lead poisoning. Dry drilling and the loading of dry muck creates infinitely more dust than wet drilling and the shoveling of muck from well-sprinkled piles. The author has repeatedly entered stopes where several drills were being operated dry and dry mucking was under way, to find the atmosphere hazy with dust even where the movement of air was fairly good. Similar stopes where wet drilling and mucking from well-sprinkled piles were in progress were found virtually free from dust. In my opinion, the use of wet drills exclusively, the thorough wetting of muck piles before they are loaded out, and adequate ventilation, will reduce to a minimum the hazard of lead poisoning in mines.

The dumping of dry ore into long chutes, where it is subjected to impact and abrasion in falling to ore pockets, also contributes to the creation of lead-laden atmospheres underground.

Lead ores are of three general classes—carbonate, sulphate, and sulphide. As has been shown previously, the three salts of lead are soluble in varying proportions in human gastric juice.

Relative solubility of lead carbonate, lead sulphate, and lead sulphide in human gastric juice at body temperature

	Percentage of solubility
Lead carbonate	46.1
Lead sulphate	9.5
Lead sulphide	2.5

From the above table it can be seen that the kind of ore being mined decidedly affects the hazard of exposure. Incidental factors other than solubility, however, are associated with these several classes of ores, as will be shown shortly.

Nearly all large deposits of lead ore in the intermountain region, especially those whose upper limits are within a few hundred feet of the surface, have a zone of carbonate ore near the top and often along the sides. The extent of this carbonate zone varies considerably from mine to mine. Many pipes or shoots of carbonate ore extend to a considerable depth.

The sulphate ores are relatively small as compared to the carbonate and sulphide and usually occur as a transition between the sulphide and the carbonate.

In most mines the carbonate zone is very dry. The carbonate ores and most of the sulphate ores are very friable; such ores break easily and produce much fine dust. This dust has a low specific gravity in comparison with that of the dust from the sulphide ore and remains suspended in the mine atmosphere for some length of time before settling. Thus, the hazard from the increased solubility of the carbonate and sulphate is increased by the occurrence of these ores in the dryer sections of the mines and by their tendency to shatter more readily and produce a lighter, finer dust.

In most mines the successive levels become more and more damp with depth until in many mines the removal of water becomes a problem. The sulphide zone is usually reached by the lower wet levels. Sulphide ore breaks in larger pieces, which, being damp, are less friable than pieces of carbonate ore and thus produce less fine dust. As sulphide ore has a high specific gravity, its dust tends to settle quickly.

Many writers have contended that lead sulphide ore is not a cause of lead poisoning in mines. It is my opinion, however, that under

certain circumstances lead sulphide may, and does, cause lead poisoning. This opinion is confirmed by several cases I have encountered. The patients gave a clear history of having worked for long periods of time in sections of mines where they were exposed only to sulphide ore. The following excerpt from Woelfel and Carlson [13] bears out this opinion:

> The following tests of the solubility of lead sulphide ore dust in human gastric juice were made at the request of Dr. Alice Hamilton, special investigator of occupational lead poisoning for the United States Bureau of Labor Statistics, who reported to us that she had found some 25 cases of lead poisoning among men employed in mines and concentrating mills of southeastern Missouri. The mines of this region are supposed to yield practically pure galena.

Galena, or lead sulphide, usually occurs as solid aggregates of crystals, but occasionally it is found disseminated as extremely fine dust, especially where sulphide ore bodies have been well underdrained and long subjected to drying and pressure. In spite of the low solubility of lead sulphide, the mining of such ore in confined spaces where there is little or no air movement can produce lead poisoning.

The incidence of lead poisoning in relation to the nature of the ore mined is illustrated in the accompanying table. The information as to the character of the ore mined was furnished by the superintendent of the mine.

Occurrence of lead poisoning and character of the ore mined in one of the larger mines of Utah for the years 1904 to 1917, inclusive

Year	Average number of employees	Cases of lead poisoning	Nature of ore
1904	375	11	Development work; carbonate ore.
1905	342	8	Carbonate ore.
1906	300	5	Do.
1907	326	4	Do.
1908	92	6	Do.
1909	183	7	Do.
1910	238	11	Do.
1911	244	8	Do.
1912	190	4	Mainly carbonate, some sulphide.
1913	193	5	Less carbonate and more sulphide.
1914	75	1	Carbonate, but larger part sulphide.
1915	106	1	Mainly sulphide ore.
1916	(1)	1	Do.
1917	(1)	0	Virtually all sulphide ore.

[1] Figures not available.

In 1910 the upper levels of another large mine in the same district were turned over to lessees. All the ore in these levels was carbonate, the ore in the lower levels, worked by company men, was sulphide. The arrangement continued for three years. The

[13] Woefel, A., and Carlson, A. J., The Solubility of Lead Sulphate Ores and of Lead Sulphide in Human Gastric Juice: Bull 141, U. S. Bureau of Labor Statistics, Feb. 17, 1914, p. 82.

table below shows the average number of men who worked in carbonate ore, the average number who worked in sulphide ore, and the number of cases of lead poisoning reported for each group.

Cases of lead poisoning among miners working carbonate and those working sulphide ores in a large mine during three years

Lessees working upper levels, in carbonate zone:	
Average number of men working	115
Cases of lead poisoning	12
Company men working lower levels, in sulphide zone:	
Average number of men working	224
Cases of lead poisoning	5

Although only about one-half as many men were working in the carbonate zone as were working in the sulphide zone, the number of cases of lead poisoning reported for the carbonate group was more than double that reported for the sulphide group.

Still more striking evidence of the relation of carbonate and sulphide ores to the incidence of lead poisoning is furnished by a comparison between the lead-poisoning experience of a district producing lead sulphide ore almost entirely and the experience of one producing highly oxidized lead ores.

Comparison between ores produced and the lead-poisoning rates for the mining districts of Bingham and Frisco, Utah, for the years 1919 to 1922, inclusive

Year	Average number of employees	Lead ore produced (short tons)	Per cent oxidized ore	Reported cases of lead poisoning	Lead poisoning rate per 1,000 employees
BINGHAM DISTRICT					
1919	1,958	109,785	0.37	2	1.0
1920	1,739	176,966	.01	0	.0
1921	1,605	157,967	.12	2	1.2
1922	1,447	91,223	3.15	1	.7
FRISCO DISTRICT					
1919	94	32,255	34.13	16	170.2
1920	66	13,805	49.23	18	272.7
1921	22	5,805	45.56	4	181.8
1922	37	9,768	44.69	3	81.0

TOXIC QUANTITIES OF THE COMMON LEAD SALTS ENCOUNTERED IN MINING

Lead, like other metals that may cause metallic poisoning, is toxic to different persons in different quantities. Even to the same person lead may prove toxic in different quantities at different times, according to the state of his general health and the normalcy of the excretory organs. When a man is physically run down and his excretory organs are sluggish he may show symptoms of lead poisoning after a light exposure, but with the same exposure, the man in

a state of good health and with his excretory organs functioning properly might show no evidence of leading.

The amount of lead necessary to cause lead poisoning in the average person has been defined by two eminent authorities, as follows:

> About 2 milligrams of lead inhaled daily represents the minimal dose which will cause plumbism. [14]
>
> A daily dose of 10 milligrams for several weeks may lead to severe acute poisoning. [15]

At first glance the amounts quoted seem somewhat at variance with each other; lead poisoning occurs in several gradations. The amount mentioned in the first statement is claimed to be the minimal dose that will cause a condition presenting symptoms, whereas the second statement gives the daily dose which will cause severe acute symptoms within a short period of time. Thus it is not hard to reconcile the difference.

As has been stated previously, most cases of industrial lead poisoning are contracted by inhaling air ladened with lead dust. In the mining of lead ores the opportunity to contract lead poisoning varies in proportion to the solubility of the ore mined, the amount of lead dust thrown into the atmosphere, and the ventilation afforded.

In most metal mines ventilation is limited; the working places are confined to relatively small areas, and because of the limited ventilation the dust thrown into the atmosphere is not dissipated as rapidly as it is in the open or in factory buildings that are adequately ventilated.

Men at labor breathe 14 to 20 liters of air per minute; that is, during a shift of eight hours they breathe approximately 7,680 liters, taking in an average of 16 liters per minute. Accepting 10 milligrams daily as the necessary amount of lead to cause lead poisoning, it can be seen that a relatively high dilution of dust from a soluble salt of lead in the air breathed may cause lead poisoning.

Assuming that a man at work breathing at a rate of 24 inhalations per minute takes in about two-thirds of a liter of air at each inhalation, then the amount of lead dust necessary in the air of each inhalation to afford ten times the minimal daily dose of 10 milligrams to cause acute lead poisoning is but one one-hundred and fifteenth of a milligram (0.000134 grain), or, by volume, 1 part in 76,800,000.

The above figures are based on a dust that is totally soluble. In mining, the common types of dust are the carbonate, sulphate, and sulphide. Based upon the theoretical amount of dust that can reach the stomach and the relative solubility of the salts mentioned, the

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[14] Legge, T. M., "Factory hygiene in 1920": Lancet (London), vol. 201, Aug. 13, 1921, p. 356.

[15] Teleky, Ludwig, Protokoll der Sitzung des grossen Rates des Instituts für Gewerbehygiene. Berlin, 1912, p. 15.

approximate quantities of each in breathed air capable of producing lead poisoning, multiplied by 10, are as follows:

Lead carbonate, one-fortieth of a milligram (0.000385 grain), 1 part by volume in 26,000,000.

Lead sulphate, one-eighth of a milligram (0.001925 grain), 1 part by volume in 5,300,000.

Lead sulphide, one-half of a milligram (0.0077 grain), 1 part by volume in 1,300,000.

From the preceding figures one can see that no great amount of lead dust need be injected into the air in mining to produce atmospheric conditions which predispose to lead poisoning.

PERSONAL FACTORS PREDISPOSING TO LEAD POISONING

Individuals differ in susceptibility to lead poisoning. Of a given number of persons exposed to the same lead hazard one group may escape poisoning entirely, while among those exhibiting evidence of lead absorption the cases may range from those that are mild, almost indiscernible, to those that are severe or even fatal.

Some of the known factors predisposing to lead poisoning are sex, age, general state of health, condition of excretory organs, hyperacidity of body fluids, personal cleanliness, and certain habits. Females appear more susceptible than males, and young adults more susceptible than persons of more mature age; lowered vitality, especially when there is interference with body excretions, markedly increases susceptibility. Hyperacidity, particularly of the gastric juice, predisposes because the lead ingested is more readily converted into soluble compounds that are easily absorbed.

Cleanliness of both the person and the clothing is an important factor in lead poisoning. Persons who do not bathe frequently or change their clothes often are exposed to a further danger from the lead dust which accumulates on their bodies and in their clothing. The clothing worn at work should not be worn at home; it should be changed for street clothing and, if possible, the body should be bathed before the change is made. Work clothes should be washed at frequent intervals to free them from lead dust. Personal cleanliness of the face and hands is likewise of much importance. Before a meal the face and hands should be thoroughly washed so as not to contaminate the food with lead dust. Drinking water should never be left open and exposed about working places where lead dust may settle in it. Containers from which drinking water is taken should have the outlets cleaned and freed from dust before they are applied to the mouth. A good practice in mining is to carry water underground in individual canteens with screw caps; with such containers not only are the contents protected but the outlet is kept free from accumulations of dust.

Personal habits play no small part in predisposing the body to lead poisoning. Time and again alcoholic intemperance has been demonstrated to be a decided predisposing causative agent. The carrying of chewing tobacco loose in the pockets of work clothes and handling it with dust-soiled hands often aids in the contracting of lead poisoning. To begin work without having previously eaten a substantial meal materially aids the absorption of lead. If a stomach is empty but has a normal supply of gastric juice, such lead as may enter is more readily converted into soluble salts easily assimilated into the system. When food is present in the stomach, the gastric juice is being utilized in natural digestion and any lead that gains entrance can be acted on only by an excess of gastric juice. Thus with a hearty meal in the stomach a good part of the lead entering the stomach may escape conversion into soluble salts. The eating of good nutritious food and the drinking of plenty of rich milk are natural aids in warding off lead poisoning. Such foods are readily digestible and when taken into the stomach combine easily with the gastric juice, thus leaving little gastric juice to act on the lead that reaches the stomach. To fail to rinse the mouth before eating or to take food in an atmosphere charged with lead dust is to encourage lead poisoning.

Lack of due regard for body evacuations also predisposes to lead poisoning. Constipation not only permits ingested lead to remain in the alimentary tract for a longer period of time, thereby increasing the opportunity for absorption, but it also interferes with one of nature's principal means of eliminating lead already in the general system. Kidney action, if neglected by failure to drink plenty of pure water, handicaps nature by slowing down one of the sources of lead elimination. All kinds of excesses that tend to lower the general health predispose to deal absorption. The failure to seek medical advice at the first sign of ill health often leads to chronic leading, which by timely action could have been avoided with little or no inconvenience. In most persons one attack of lead poisoning predisposes to other attacks on reexposure. With each subsequent attack the tolerance of the body for lead seems to grow less and less. A probable cause for this is that certain tissues of the body develop a hypersensitiveness to lead, and a complete elimination of lead from the system is seldom effected; hence, the added quantity sufficient to produce symptoms is less than the amount originally required.

Men engaged in mining who contract well-marked cases of lead poisoning should either give up mining or seek employment in mines free from lead.

ONSET OF LEAD POISONING

Lead is a cumulative poison. A large amount may be taken in a single dose and yet the patient will exhibit very slight effects. The

reason for this is that the system can absorb only a limited quantity at a time, and that portion not acted upon, which is the bulk in large doses, is expelled in the feces. Virtually all authorities agree that plumbism is much more likely to result and the symptoms to be more pronounced when small quantities of lead continually enter the body during a long period of time. The small doses ingested are absorbed and the amount within the system is gradually built up to a point where functional derangements result from the accumulated poison.

There is a distinction, however, between lead absorption and lead poisoning. When lead is absorbed into the system it is taken into the general circulation and carried to the various organs and tissues of the body. Nature immediately attempts to counteract absorption by eliminating lead through certain excretory channels, such as the liver, gall bladder, and bowels, the kidneys and urinary tract, and to a limited extent, the skin. Where the rate of absorption is greater than the rate of elimination the lead stores up in certain tissues and gives rise to the symptoms characteristic of lead poisoning.

As has already been pointed out under "Factors Predisposing to Lead Poisoning," inherent conditions, peculiar to different persons—such as age, sex, body conditions, and habits—to a large extent control both the opportunity for exposure and the rate of absorption. Similar inherent factors peculiar to different individuals likewise influence elimination. The variance in these factors in indviduals no doubt accounts for the variance in length of exposure necessary to produce symptoms of leading.

The following tables, giving the periods of exposure to lead before the onset of the symptoms of lead poisoning occurred, furnishes some idea of the variance of susceptibility in different persons. No statistics were available for mining experience, so that use was made of cases which occurred in other industries where the workers are exposed to lead.

Period of exposure to lead before onset of symptoms of lead poisoning in the manufacture of storage batteries in the United States[16]

	Cases
Less than 1 month	6
1 month and less than 2 months	11
2 months and less than 3 months	14
3 months and less than 4 months	12
4 months and less than 5 months	5
5 months and less than 6 months	2
Total less than 6 months	50

[16] Hamilton, Alice, Lead Poisoning in the Manufacture of Storage Batteries: Bull. 105, U. S. Bureau of Labor Statistics, December, 1914, p. 26.

	Cases
6 months and less than 7 months	2
7 months and less than 8 months	2
8 months and less than 9 months	0
9 months and less than 10 months	2
10 months and less than 11 months	0
11 months and less than 12 months	2
Total less than 1 year	8
1 to 2 years	1
4 to 5 years	1
Total over 1 year	2
Grand total	60

Length of exposure of white lead workers previous to onset of lead poisoning[17]

Less than 1 week	1
1 week and under 2 weeks	7
2 weeks and under 3 weeks	16
3 weeks and under 4 weeks	12
1 month and under 2 months	25
2 months and under 3 months	7
3 months and under 4 months	6
4 months and under 5 months	5
5 months and under 6 months	3
Total, less than 6 months	82
6 months and under 7 months	4
7 months and under 8 months	1
8 months and under 9 months	0
9 months and under 10 months	2
10 months and under 11 months	0
11 months and under 12 months	0
Total, less than 1 year	7
1 year and under 2 years	13
2 years and under 3 years	6
3 years and under 4 years	3
4 years and under 5 years	2
5 years and under 6 years	2
6 years and under 7 years	2
7 years and under 8 years	0
8 years and under 9 years	0
9 years and under 10 years	1
Over 10 years	2
Total, over 1 year	31
Grand total	120

[17] Hamilton, Alice, The White-Lead Industry in the United States, with an Appendix on the Lead-Oxide Industry: Bull. 95, U. S. Bureau of Labor Statistics, July, 1911, p. 224.

Period of exposure of smelter and refinery employees before onset of lead poisoning [18]

	Cases
Less than 1 month	18
1 month and less than 2 months	19
2 months and less than 3 months	22
3 months and less than 4 months	33
4 months and less than 5 months	17
5 months and less than 6 months	12
6 months and less than 12 months	11
Total less than 1 year	132
1 year and less than 2 years	16
2 years and less than 3 years	8
3 years and less than 4 years	4
4 years and less than 5 years	1
5 years and less than 6 years	2
6 years and less than 7 years	0
7 years and less than 8 years	3
8 years and less than 9 years	0
9 years and less than 10 years	0
Over 10 years	1
Total over 1 year	35
Grand total	167

A striking fact shown in the three preceding tables is the high incidence of lead poisoning occurring after less than six months of exposure. The relative percentages of these cases for the three tables are 83 per cent, 68 per cent, and 72 per cent, respectively. The cases occurring after an exposure of less than four months number more than half of the total cases in each table. The percentages for this period are as follows: Storage battery workers, 71 per cent; white-lead workers, 61 per cent; and smelter and refinery workers, 55 per cent.

The insidious tissue alterations and lesions in lead poisoning take place so gradually and the resulting symptoms manifest themselves in such mildly increasing severity that frequently those suffering from lead absorption little suspect the true cause of their ailment until well-defined cases of lead poisoning have developed. The character of its symptoms—the gradual lowering of the general health, an increasing pallor and muscular weakness—and the absence of acute symptoms tending to point to a definite cause no doubt are reasons why lead poisoning has prevailed to the extent that it has.

SYMPTOMS OF LEAD POISONING

The early symptoms of lead poisoning are usually ill-defined, and because of their vagueness they are apt to be overlooked. The most

[18] Hamilton, Alice, Lead Poisoning in the Smelting and Refining of Lead: Bull. 141, U. S. Bureau of Labor Statistics, February, 1914, p. 64.

usual symptoms, in the order in which they become manifest, are as follows:

Symptoms of incipient lead poisoning

Lassitude,
Slowly increasing pallor,
Vague gastric uneasiness,
Disagreeable sweetish metallic taste,
Decrease in appetite,
Oppression after eating,
Headache,
Persistent loss of strength and weight,
Mild anæmia,
Constipation, or alternating constipation and diarrhea.

Any of the above symptoms may be entirely absent and others may be present in an aggravated form.

In well-defined or acute cases the symptoms in the list just given are usually most severe. The anæmia is more marked, with a red cell count between 2,500,000 and 3,500,000 per cubic millimeter, and there is a corresponding diminution of the hæmoglobin; structural changes in the red cells of the blood progress from anisocytosis to polychromasia, and basophile granulations are frequently noted. The pallor is increased out of proportion to the red cell count and the hæmoglobin index. Lassitude has given way to marked muscular weakness. The gastric uneasiness takes the form of severe abdominal pains, frequently colicky, that originate in the vicinity of the umbilicus and radiate in all directions. The abdominal muscles are usually tense, and the entire abdomen is tender. Vomiting may or may not take place. Obstinate constipation usually prevails, and exceedingly strong medication is necessary to secure evacuation of the bowels. Bowel movement in most cases does not permanently relieve the abdominal pains.

Marked loss of appetite is usual; the secretion of saliva is decreased and the breath is fetid. Along the gingival margin the gums often present a grayish or blue line, most marked in the neighborhood of the upper canines, due to the deposition of lead sulphide in the walls of the superficial capillaries.

Headache is present in a considerable number of cases; it is often severe, but not confined to any one part of the head. Tremors of the tongue and hands may or may not be present. The labiofacial muscles also may show tremors. Ill-defined arthritic pains in the limbs, particularly in the region of joints, due to local patches of neuritis, are often noted. Slight rises of temperature are not infrequent, and the patient may complain of defects of vision.

The blood pressure is increased and lack of nervous stability is noticeable. Depression, confused cerebration, and defective memory are often noted in severe cases.

Chronic lead poisoning may manifest itself by a variety of symptoms by which, to a certain extent, the cases of chronic lead poisoning are classified into the following three general groups:

1. Constitutional group, which includes the bulk of the cases. This group presents mainly symptoms of glandular and visceral involvement, with only moderate symptoms of derangement of the nervous system.

2. Cerebral group, primarily involving the central nervous system; in this group the most marked symptoms originate within the brain.

3. Peripherospinal group, primarily involving the central nervous system; in this group the most marked symptoms originate below the cerebrum.

Lead, after having been taken into the system, is circulated through the vascular system and execreted through certain organs. When lead is ingested over a considerable period of time certain tissues with which it comes in contact undergo a loss of normal tonicity and in some cases even suffer degenerative changes. It is the results of the changes mentioned that bring about those cases typical of the constitutional group of chronic lead poisoning.

Where the amount of lead ingested is greater than that which the excretory systems can eliminate the excess is stored within the body. Recent studies by Minot,[19] Minot and Aub,[20] and Fairhall and Shaw, [21] prove almost conclusively that most of the lead found in the system is stored within the skeleton. The excess lead remaining in the general circulation as a result of the failure of the excretory organs to eliminate all the metal ingested is carried to the various organs of the body. Although the bones show a special affinity for lead in the blood and are capable of immobilizing it through storage in the skeleton structure, much damage to delicate tissues is done by the circulating lead before it reaches the bones. Lead exhibits a predilection for nervous tissue and in certain subjects this predilection is greater than in others. The toxic effect of lead on nerve tissue seems to be selective even as to location. If through selective action the circulating lead produces pathologic lesions within the brain tissues, naturally the toxic effects will be manifested by symptoms which originate within the brain, producing cases classified under the cerebral group. Where the selective action is confined chiefly to the nerve tissue of the spinal cord and peripheral nerves, the symptoms produced are those of the peripherospinal group.

In chronic lead poisoning those symptoms already mentioned as characteristic of acute lead poisoning may be present in part or in

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[19] Minot, A. S., "The distribution of lead in the organism after absorption by the gastrointestinal tract": Jour. Ind. Hygiene, vol. 6, August, 1924, p. 125. "The distribution of lead in the organism after absorption by the lung and subcutaneous tissue": Jour. Ind. Hygiene, vol. 6, August, 1924, p. 137.

[20] Minot, A. S., and Aub, J. C., "The distribution of lead in the human organism": Jour. Ind. Hygiene, vol. 6, August, 1924, p. 149.

[21] Fairhall, L. T., and Shaw, C. P., "The deposition of lead salts, with a note on the solubility of di-lead phosphate in water at 25° C. and tri-lead phosphates in lactic acid at 25° C.": Jour. Ind. Hygiene, vol. 6, August, 1924, p. 159.

whole and may be severe or moderate in intensity. Among the symptoms most common to the several groups of chronic lead poisoning the following deserve mention:

Symptoms of chronic lead poisoning

Constitutional group:
- Almost any of the vital structures may exhibit degenerative changes.
- Gastrointestinal disturbances.
- Lead colic.
- Atrophic degeneration of the intestinal glands and walls.
- Obstinate constipation.
- Albuminuria.
- Cloudy swelling of the kidneys.
- Interstitial nephritis.
- Fibrosis of the kidneys.
- Arthralgia, simulating gout.
- Arteriosclerosis.
- Cirrhosis of the liver.

Cerebral group:
- Onset often sudden and of great violence. May take the form of maniacal delirium, or stupor. Often preceded by headache, giddiness, sleeplessness, and disturbances of the organs of the special senses.
- Severe headache.
- Defective memory.
- Inequality of pupils.
- Retinal changes.
- Loss of vision.
- Saturnine encephalopathy.
- Vomiting of centric origin.
- Cerebal hemorrhage.
- Poliencephalitis.

Peripherospinal group:
- Lead palsy, tremors of the tongue, hands, and labiofacial muscles.
- Paralysis, wrist drop, usually bilateral, ankle drop; facial paralysis occasionally occurs.
- Interstitial neuritis, chiefly in limbs and joints.
- Uneven gait in locomotion.
- Chorea.
- Dysponea.
- Paralysis of rectum and bladder.
- Poliomyelitis.

The symptoms in many cases of chronic lead poisoning do not conform to any one of the above groups but are distributed among all three groups.

Certain statistics gathered under the factory and workshop act of England as reported by Dr. Thomas M. Legge [22] furnish data on the severity of cases, number of attacks, and the main symptoms in 9,380 cases of lead poisoning reported during the years 1900 to 1914.

[22] Legge, T. M., "Twenty years' experience of the notification of industrial diseases": Jour. Ind. Hygiene, vol. 1, April, 1920, p. 590 et seq.

Classification of characteristics of the cases of lead poisoning reported in England, 1900–1914

	Severe	Moderate	Slight	Not stated	Total
Severity of attack:					
Cases	2,269	2,565	4,343	203	9,380
Per cent	24.2	27.3	46.3	2.2	100

	First	Second	Chronic	Not stated	Total
Number of attacks:					
Cases	6,722	1,312	1,063	283	9,380
Per cent	71.7	14.0	11.3	3.0	100

	Gastric	Anemia	Head-ache	Paretic	Enceph-alopathy	Rheu-matic	Other
Main symptoms:							
Cases	7,369	2,735	1,178	1,691	325	996	390
Per cent	78.5	29.2	12.6	18.0	3.5	10.6	4.2

In the table of main symptoms the total of the percentages for the several symptoms amounts to considerably more than 100 per cent, due to the fact that some cases presented main symptoms belonging to more than one group.

DIAGNOSIS OF LEAD POISONING

The diagnosis of lead poisoning is made with little difficulty when the case has progressed to a stage where well-marked symptoms, both objective and subjective, present themselves. Those cases where elimination is only slightly below absorption and the lead circulating within the system fails to make its presence apparent through well-defined symptoms are the ones that are hard to diagnose. It is in order to avoid permanent disabilities and to prevent passing to the acute or chronic stages that the physician by an early diagnosis, even if it is only provisional, can accomplish the most good.

In lead mining, as in other industries that involve exposure to lead, a history of the employment should at once arouse a suspicion as to the probable cause of the ailment. In the absence of other well-defined causes for the indisposition, lead should be accepted as the cause. By closely questioning the patient as to the progress of his case, weighing all the subjective symptoms and clinical findings, and following the case for even a few days, the physician usually obtains evidence that lead is the exciting cause.

For diagnostic purposes the findings are divided into two groups—subjective symptoms as derived from the history of the case furnished by the patient; objective symptoms as determined through examinations.

Subjective symptoms in the usual order noted by patient

History of exposure to lead.

Pallor that has gradually increased.

Lassitude passing to debility and general muscular weakness.

Sweetish metallic taste, especially in the morning on arising.

Decreasing appetite, particularly for the morning meal.

Frequent nausea after the morning meal.

(The above two complaints are invariably present and are peculiar to several metallic poisonings.)

Persistent loss of weight.

Depression.

Constipation usually, but may alternate with diarrhea.

Gastrointestinal disturbances, general feeling of abdominal uneasiness, which later manifests itself in the form of severe cramps.

Headache, becoming more and more frequent and increasing in severity.

Nervousness.

Vertigo.

Insomnia.

Darting fugitive pains in limbs, becoming arthritic in character.

Ringing in the ears.

Defective vision, spots before the eyes, blurring and double vision.

Lack of tonicity of extensor muscles of fingers and wrist, later followed by wrist drop.

Objective symptoms that are not always present in their entirety

Pallor.

Emaciation, with noticeable loss of subcutaneous fat of the infraorbital regions.

Mental confusion.

Impaired memory.

Fetid breath.

Gums ulcerated at margin of teeth.

Blue line on gums, present in about 20 per cent of cases.

Tremor of tongue.

Tenderness of muscles in the region of the joints, with tingling sensations.

Tremors of hands, and inability to grasp firmly.

Anesthesia or parasthesia, especially of the arms.

Abdominal distention and tenderness.

Cramps with pains radiating from the umbilicus, cramps recurrent and not relieved by evacuation of the bowels.

Obstinate constipation responding only to very strong purgatives. Constipation may alternate with diarrhea. (The above three symptoms, taken together with a reasonable number of subjective symptoms, are characteristic of lead poisoning.)

Increased blood pressure, with palpitation.

Arterioclerosis.

Blood findings not compatible with degree of pallor.

Blood thin and watery.

Red-cell count reduced to between two and one-half to three million per cubic millimeter, with a corresponding drop in hæmoglobin. Anisocytosis and polychromiasis. Basophilic degeneration of red cells is present in a considerable number of cases ranging in extent from only a few to several hundred per

million red cells. By some authorities it has been claimed that the presence of punctate cells is pathognomonic of lead poisoning, and their presence was considered one of the earliest signs of lead intoxication. Subsequent investigations tend to show that punctate cells may be so few as to escape notice in well-marked cases of lead poisoning, and also they may be present in pathological conditions where there is no possibility that lead is the causative agent. Their presence, however, with a history of exposure to lead and any probable sign of lead poisoning, should be accepted as very presumptive evidence.

Incontinence, urine decreased in quantity, with nocturnal frequency.

Albuminuria.

Where the symptoms in the foregoing list are not present in sufficient numbers or severity for the physician to make a diagnosis of lead poisoning, the presence of eliminated lead in the urine or stools can, the author believes, be accepted as positive evidence of lead intoxication.

BLOOD TEST

In a few industrial plants, where it is known that danger from exposure to lead exists, the plan of making periodic blood examinations has been put into practice in order that cases of absorption may be determined early before definite symptoms of poisoning can develop. For large groups of employees such a method seems to give satisfactory results.

Although it is not claimed that a blood test is an infallible index, yet the ease with which smears from large numbers of employees can be taken and examined and the relative reliability of the results obtained makes the method the most practical yet devised as a check on individual absorption.

In a large lead refinery and white-lead plant the author recently had an opportunity to study the application of and the results obtained through a system for detecting lead absorption by the examination of blood smears. The method of detecting lead absorption was developed by the late Dr. G. W. Miller, formerly the plant physician. The main value of the method is that it detects the gradual changes in the blood corpuscles as increasing amounts of lead are absorbed. The range of these particular tests covers the entire period between normality and the stage at which the individual is incapacitated for work.

Prior to the use of this means of detection, the first indications of absorption were the usual vomiting, constipation, or diarrhea, and loss of appetite. By the blood tests these symptoms appear as the last stages and correspond to general saturation of the corpuscles.

The sample of blood for the test is drawn from the lobe of the ear. The surface of the skin is sterilized, pierced, and a drop of blood squeezed out and received on a sterilized microscopic slide. The drop

is spread over the slide by drawing across it the edge of a second slide. The two slides are then placed together in a holder and marked with the number of the individual. The smears are examined by a pathologist, and his findings are submitted to the plant physician who makes the diagnosis.

The stages indicated by the blood-smear tests, listed in the order of their occurrence, and a few of the characteristic symptoms of each stage, are as follows:

Stages indicated by blood-smear tests

	Pathologist's report	Characteristic symptoms
Normal stage	Negative	None.
First stage	Anæmia	Slight lowering of the vitality.
Second stage	Anisocytosis and anæmia	First rearrangement of blood cells. Rare cases show occasional vomiting; average case feels no discomfort.
Third stage	Polychromasia with anisocytosis	Complete rearrangement of cells and change in coloring; occasional cramps and stomach disorders; patient inclined to be irritable.
Fourth stage	Mild positive. Few stipple cells; polychromasia and anisocytosis.	Granules within cells; general distortion of cells; patient dull and listless; noticeable decrease in efficiency; irritable; complains of constipation or diarrhea.
Fifth stage	Positive; numerous stipple cells; polychromasia and anisocytosis.	Acute stage; vomiting, constipation, or diarrhea; colic, tongue coated, and blue line on gums.

The length of the time interval between the various stages depends upon the severeness of the exposure, and the preventive care exercised by the person exposed. The two following representative records of employees may be of interest:

The employee's record which follows represents the gradual development and variation in the tests made between October, 1921, and February, 1922. This employee had worked in the plant since 1919, but the examination of blood smears was not inaugurated until October, 1921. He worked continuously during the period covered by the tests, and the nature of his work was regulated by the findings of the current tests made.

Date of test and pathologist's report:

October 18, 1921. Anæmic, hyaline.
November 15, 1921. Negative.
December 12, 1921. Positive; stipple cells.
December 27, 1921. Mildly positive; few stipple cells; anisocytosis.
January 13, 1922. Very mildly positive; few basophilic red cells.
January 26, 1922. Positive; stipple cells; polychromasia.
February 14, 1922. Mildly positive; few stipple cells.
February 24, 1922. Mildly positive; few stipple cells.

The following record is that of an employee who has been continuously employed in a department where careful personal hygiene to avoid absorption is required.

Date of test and pathologist's report:

October 18, 1921. Mild anæmia.
November 15, 1921. Negative.
December 12, 1921. Negative.
December 27, 1921. Very mildly positive; occasional stipple cell.

Date of test and pathologist's report—Continued.
January 13, 1922. Negative.
January 26, 1922. Negative.
February 14, 1922. Negative.
February 24, 1922. Negative.

Since the time that the periodic examination of blood smears was begun at this plant in October, 1921, monthly examinations have been made of all the employees in the white-lead department, except during several periods when the plant was not running. A total of 1,271 examinations had been made when the data were obtained in September, 1924. The following are the findings given by these examinations:

Negative	941
Negative plus	30
Very mildly positive	69
Mildly positive	140
Positive	91

In 1924 the plan of examination by blood smears was extended to include all other employees of the lead refinery, who accordingly were examined once every three months. A total of 211 examinations had been made by September, with the following results:

Negative	164
Negative plus	5
Very mildly positive	7
Mildly positive	20
Positive	15

After each blood-smear examination, the practice at this plant is to supply medicine through the safety engineer to all employees who show evidence of lead absorption. Cases showing mildly positive and positive tests report to the plant physician, and after close examination the plant physician and safety engineer determine the advisability of transferring these employees to a class of work where the lead hazard is less.

It is not claimed that blood-smear examinations furnish an absolute index to lead absorption, but the experience at this plant has been that only rarely have cases showing clinical signs of lead absorption furnished negative blood smears.

For the purpose of checking the results obtained in preventing disabilities from lead poisoning the records of all employees in the white-lead department for the four months preceding the author's visit were examined. During this period the white-lead department employed an average of 54 men, and despite the known hazards in the manufacture of white lead there was not a single day lost on account of sickness.

In chronic cases of lead poisoning such symptoms as well-marked wrist drop, persistent lead colic, or encephalopathy, with a history

of exposure to lead, should clearly indicate to the attending physician the nature of the illness. In long-standing cases where no exposure has taken place for months it is not always possible to detect lead in the urine or feces.

Gowers's three postulates for the medicolegal test for a positive diagnosis of plumbism are given as follows:

1. Lead must be demonstrated as entering the body.
2. Indisputable clinical symptoms of lead poisoning must be demonstrated, showing that lead is within the body.
3. Demonstration of the excretion of lead from the body.

With respect to medicolegal considerations, the fulfillment of the requirements of the above postulates would probably be necessary, but if in the diagnosis and treatment of industrial lead poisoning a patient gives a history of exposure and presents a sufficient number of subjective and objective symptoms to arouse a suspicion that lead is the cause, the attending physician is derelict in his professional duty if he does not immediately take steps to prevent further exposure and institute the proper expectant treatment to eliminate such lead as has already been stored within the system. The progress of the case under such a régime will soon show whether or not lead is the causative agent.

TREATMENT OF LEAD POISONING

The measure of first importance in the treatment of lead poisoning is to remove the patient from further exposure to lead absorption. In mild cases where the elimination rate is only slightly below the rate of absorption and an early diagnosis has been made, the prevention of further opportunity for absorption permits the system to rid itself of lead with little or no medical interference. By placing such patients in a changed working environment free from exposure to lead, preferably in the open air; providing a diet of nourishing and easily digested food, including an abundance of milk; mild tonics; an adequate amount of rest; frequent warm baths; and careful regulation of the excretions, particularly the bowels, the mild cases will usually be cleared up in a comparatively short time.

In the acute cases the first consideration is the palliative treatment of the predominant symptoms and the promotion of lead elimination through the proper hygienic régime and medication.

Constipation, depending upon its persistence, may be relieved by simple enemas, salines (of which magnesium sulphate is highly recommended under the supposition that lead present in the intestines is converted into less soluble lead sulphide), castor oil, and in extreme cases croton oil given in 1 minim dose on a lump of sugar.

Lead colic may be treated according to its severity by keeping the patient in bed, by carminatives, by application of heat to the abdomen, and by opiates. Relieving the constipation and keeping the bowels open frequently have a markedly beneficial effect in preventing the recurrence of paroxysms of colic.

The treatment of headache caused by plumbism is frequently far from satisfactory. Some cases respond to the usual migraine remedies, while others can only be relieved through the use of somnifacients or opiates. However, where further exposure to lead has been prevented and elimination has been stimulated, the cerebral symptoms in acute cases usually subside with marked rapidity.

The gastric disturbances readily clear up with the patient's removal from further danger of ingesting lead, and with proper hygienic regulation and careful selection of diet. The hygienic procedures of first importance are frequent warm baths, cleanliness of the mouth by use of alkaline mouth washes, suitable warm clothing, especially about the abdomen, if the patient is up and around, and the regulation of the bowels. The diet should consists of easily digested foods containing readily assimilable fats, rich milk, rich broths, and plenty of butter. Food should be taken at frequent intervals and in small quantities at first, so that the digestive processes may be gradually restored to normal condition.

Lassitude, asthenia, and anemia are best treated by plenty of rest and sleep and general tonics. Quinine, strychnine, and iron, either as elixir or in tablet form, form one of the best tonics for lead poisoning. The quinine acts as a general alterative and mild stomachic; the strychnine as a digestive, cardiac tonic and nerve tonic; and the iron as a hematic tonic.

Neuritic pains, if present, may be relieved by hot applications, electricity, and massage.

Chronic cases resolve themselves largely into cases demanding, in addition to the general treatment outlined above, the relief or correction of conditions due to degenerative or atrophic changes set up in the nervous, digestive, vascular, or genitourinary systems. One or more of these systems may be involved. The degenerative or atrophic changes in many cases will not have proceeded to such a stage that they are beyond correction.

Besides assisting lead elimination and building up the general condition of the patient as much as possible, the usual procedure is the application of expectant treatment of the chronic symptoms exhibited.

At different times several specific forms of treatment have been recommended for lead poisoning, among which may be mentioned the following:

Potassium iodide has been claimed to assist materially in the elimination of lead. Several authorities, however, from their experience in the treatment of chronic cases of lead poisoning, have shown that the use of potassium iodide may cause a sudden release of stored lead in such quantities as to cause a return of acute symptoms. This stored lead when thrown into the general circulation in a form capable of conversion into absorbable lead compounds excites exacerbations of serious acute symptoms because of the reabsorption of the lead before it can be eliminated from the system.

The Clague-Oliver electrolytic treatment for lead poisoning has been given many trials both with animals and human beings; but, except in the rabbit experimented with by Sir Thomas Oliver, this system of treatment has practically never proved efficacious in either the elimination of lead from the body or the permanent relief of symptoms of lead poisoning.

Two investigations of the efficiency in the treatment with the Clague-Oliver electrolytic process of cases of lead poisoning among pottery workers were made by the Office of Industrial Hygiene and Sanitation of the United States Public Health Service. A summary of the findings in these two investigations tends to show that although temporary relief from certain subjective symptoms appeared to be effected in some of the cases, these same cases according to the diagnosis of the examiner showed a slowly progressive plumbism. Other cases showed no physical improvement; in fact, in some of them the intoxication slowly progressed in spite of the treatments.

TREATMENT WITH SODIUM THIOSULPHATE

Sodium thiosulphate is by no means a new remedy, although its application in the treatment of lead poisoning has been recognized only within the last few years. Sodium hyposulphite had been used before 1911 as a preventive for lead poisoning with apparently marked success, as cited by Oliver:[23]

> In one of the best conducted white-lead works with which the writer is familiar, the firm provided chocolate-coated tabloids containing 5 grains of hyposulphite of soda. The men of their own accord take sometimes two or three of these tabloids daily, and although on the gums of some of the men there is a well-marked blue line, and in their urine lead has been found, yet in none of the men are there symptoms of lead poisoning.

Several investigators in this country have used sodium thiosulphate in the treatment of lead poisoning. Notable among them are Dr. Nelse F. Ockerblad of Kansas City, Kans.; Dr. George F. Roberts and Dr. Andrew J. Hosmer, of Salt Lake City, Utah; and Dr. C. S. Nelson and Dr. L. S. Milne, of Kansas City, Mo. In every case on

[23] Oliver, Thomas, Industrial Lead Poisoning, with Descriptions of Lead Processes in Certain Industries in Great Britain and the Western States of Europe; Bull. 95, U. S. Bureau of Labor Statistics, July, 1911, p. 139.

which I have been able to secure data where sodium thiosulphate has been used in the treatment of poisoning from lead, the results have been uniformly succesful. To date, however, the number of cases treated has been too few to warrant the conclusion that sodium thiosulphate is a specific for lead intoxication, but the results so far obtained are sufficiently encouraging to stimulate further and complete investigation of the application of that remedy in lead poisoning.

A few of the cases in which sodium thiosulphate treatment has been applied, the methods of administration, and the results obtained are cited.

Case 1. W. D., a white male, aged 47; dispensary No. 28206, presented himself at the urological clinic July 28, 1922, for relief of a chronic urethral discharge. * * *. He was treated in the routine manner until September 25, when he was given a prescription containing zinc sulphate and lead acetate to use morning and night as a urethral injection. The 4-ounce mixture contained of zinc sulphate gr. viii, of lead acetate gr. x. The patient professed to understand the instructions perfectly and repeated in English his understanding of the matter, which was essentially correct. He was next seen on October 9, at which time he had profuse hæmaturia. On cystoscopy blood was seen coming from both ureters. On October 11 he had developed pain in the epigastrium and a malaise with still more hæmaturia. Upon checking up on the case it was found that the patient had consumed two 4-ounce bottles of the above mixture, taking it internally three times a day. He had pallor and a lead line on his gums. The obvious conclusion was that this man was suffering from lead poisoning from having taken by mistake a medicine containing lead acetate and intended for application to the urethra. He was sent to the hospital for treatment and observation. The blood examination showed characteristic stippling of the red cells commonly accompanying the toxemia produced by lead. He had casts and albumin in his urine in addition to blood. His red blood count was 4,288,000; 75 per cent hæmoglobin; 7,000 white blood cells. He was immediately given daily injections of 0.6 gram of purified hyposulphate of soda, intravenously. On the second day the urinary hæmorrhage ceased and there was no return of it. The patient was kept on hyposulphite injections as above for two weeks. He made a good recovery, is free from symptoms of lead poisoning, and has returned to work.[24]

Case 2. White-lead painter, 60 years of age, who for the past 28 years, during which time he has worked as a painter, had suffered more or less with colic. He was in a wheel chair when the doctor first saw him and had been confined to that chair for two years. He had acute neuritis in both sciatic nerves, with general arteriosclerosis. One gram of sodium thiosulphate was given intravenously three times a day for two weeks. Result: Immediate relief from pain; improved appetite; general improvement in condition; he abandoned his wheel chair and is walking with a cane.[25]

Case 3. J. S., Serbian, aged 40, had been away from duty for one week on account of abdominal pain. His family physician had used all the better-known remedies for lead poisoning with practically no effect. At the beginning of the second week he applied to us for treatment. The first day he was

[24] Ockerblad, N. F., "A case of hematuria from lead poisoning": Jour. Urology, vol. 10, September, 1923, pp. 273 and 274.

[25] Milne, L. S., Unpublished case, Kansas City, Mo.

given 1.2, 1.6, and 1.8 grams of sodium thiosulphate intravenously. The second day the dosage was repeated. By the third day his pain had all disappeared. The third day he was given two doses of 1.6 and 1.8 grams each. The fifth day he returned to work a well man and has not applied for any treatment since that date.[26]

Case 4. R. J., negro plumber, aged 32. For two months he had been practically incapacitated on account of abdominal pain. One month of this time had been spent in hospitals. On admission to the county hospital he was in such pain that his thighs were almost completely flexed on his abdomen. His bowels had not moved for four days. He was given one-half grain of morphine, 2 ounces of magnesium sulphate, and three doses of 1.6 grams each of sodium thiosulphate intravenously during the first 24 hours. The next day his pain was greatly relieved. The sodium thiosulphate was again repeated. The second day his pain had practically disappeared. The third, fourth, and fifth days he was given two doses each of 1.2 grams. By the sixth day his pain had all gone and he left the hospital to return to work.[26]

The medical service of one of the large smelters near Salt Lake City has been using sodium thiosulphate for more than a year with excellent results in the treatment of cases assumed to arise from exposure to lead. It has become the routine procedure to give intravenous doses of sodium thiosulphate, even before their blood shows any change, to all the men applying for treatment who complain chiefly of vague abdominal pains or indefinite symptoms simulating those due to lead absorption. After several treatments the symptoms are entirely cleared up. Dr. Andrew J. Hosmer, Dr. J. S. Alley, and Dr. R. W. Quick, of Midvale, Utah, have furnished the author with the records of 28 such cases, ranging from those so mild that the men continued at work, to old chronic cases of patients so incapacitated that they could not work more than half time. Two cases had developed wrist drop. The number of intravenous injections given ranged from one to six. Eight cases received one treatment, 3 cases received two treatments, 1 case received three treatments, 1 case received four treatments, and 15 cases received six treatments. All of these cases made good recoveries; all symptoms of lead poisoning were cleared up, and not a single incidence was recorded where the use of sodium thiosulphate produced any toxic or other ill effect.

From the experience of those who have used sodium thiosulphate in the treatment of lead poisoning the following conclusions have been drawn: When given intravenously sodium thiosulphate rapidly relieves the distress incident to lead poisonings and apparently hastens the elimination of the toxic properties due to the poisonings; sodium thiosulphate may be given in fairly large doses intravenously at frequent intervals with nontoxic effect.

[26] Roberts, G. F., and Hosmer, A. J., Use of Sodium Thiosulphate in Metallic Poisonings: read before the Salt Lake County Medical Society, Apr. 12, 1924.

PREVENTION OF LEAD POISONING IN THE MINING OF LEAD ORES

The prevention of lead poisoning in mining resolves itself into two separate but allied sets of responsibilities, those devolving upon the company and those devolving upon the employees.

The company responsibilities are primarily to maintain working conditions in which the lead hazard is reduced to the lowest degree possible by observing the following precautions:

Underground drilling to be wet.

Piping an adequate supply of water to all working faces.

Sprinkling muck piles thoroughly before loading.

Blasting only at end of shift.

Adequately ventilating every working place in the mine.

The company responsibilities not only require that facilities be provided for carrying out these precautions but that through strict supervision proper uses be made of these facilities by employees, so as to keep the mine atmosphere as free from dust as is possible.

Having reduced the lead hazard to the lowest possible point at its source, the company has the further responsibility of providing measures such as the following to offset any exposure to lead as may still occur: A good change house having plenty of hot and cold water, with an average of one shower to every 10 employees; a good medical service, including periodic examinations of all employees by which men showing the least suspicion of lead absorption can be placed under observation and treatment at the earliest possible time; shifting men showing suspicious signs of lead absorption to sections of the mine with less lead hazard, or to surface work; and education of employees as to the ways and means of warding off lead poisoning.

The responsibilities of the employees are, first, cordial cooperation with the company in its efforts to limit the lead hazard underground; and, second, the practice of certain rules of personal hygiene and habits, including:

Keep body clean.

Bathe at end of each shift and change to street clothing.

Change and wash work clothes frequently.

Wash hands before eating.

Never eat food in a dusty atmosphere.

Rinse mouth before eating.

Eat a substantial meal before reporting for work.

Eat plenty of nutritious food.

Keep body excretions free. Do not permit yourself to become constipated.

Avoid all excesses, particularly alcoholic beverages.

Consult company physician at first sign of ill health and report for periodic examinations regardless of how well you feel.

CONCLUSIONS

Lead poisoning contracted in the mining of lead ores is much more common than has been believed.

Virtually all lead poisoning at mines is contracted through inhaled lead dust.

The carbonate ores are the most frequent cause of lead poisoning among miners.

The sulphide ores may cause lead poisoning when mined dry in poorly ventilated places.

Lead poisoning in mining can be reduced to a minimum by efficient ventilation, wet drilling, and the sprinkling of muck piles before loading.

Mine employees have an equal responsibility with the company in preventing lead poisoning.

Proper medical supervision will do much to prevent the development of the chronic cases of lead poisoning common in the past.

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